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EFFECT OF PARENTAL CIGARETTE SMOKING ON THE PULMONARY FUNCTION OF CHILDREN

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The authors have investigated the effects of parental smoking patterns on the pulmonary function of children in East Boston, Massachusetts. A crude inverse dose-response relationship was observed between the level of FEF_{25-75}% predicted of children who never smoked and the number of smoking parents in the household. Compared to children with two non-smoking parents, the level of FEF_{25-75}% predicted was 0.156 and 0.355 standard deviation units lower for children with one and two currently smoking parents, respectively. An additional decline in level of FEF_{25-75}% predicted was observed for children who themselves had smoked. Smoking children with two smoking parents had an average FEF_{25-75}% predicted level which was 0.355 standard deviation units lower than non-smoking children with two smoking parents. These data not only confirm that cigarette smoking by young children and teenagers has direct measurable effects on their pulmonary function, but also show that cigarette smoking by parents has a measurable effect on the pulmonary function of their children which is independent of any direct use of cigarettes by the children.

respiratory tract infections; smoking

Despite the continued warnings concerning the health hazards of cigarette smoking, large numbers of adults continue to smoke cigarettes (1), and increasing numbers of teenagers have also taken up the practice (2). The continued use of cigarettes has raised concerns regarding the effect on non-smokers of breathing cigarette smoke. A number of studies (3) have documented that, depending on such factors as ventilation, the number of cigarettes smoked, and the volume of the enclosed space, cigarette smoking can impose a pollutant burden that may potentially be hazardous to the health of exposed non-smokers.

Other studies (4–11) have demonstrated that children who are exposed to parents who smoke carry an increased burden of respiratory illness. This association has been observed as early as the first year of life (7, 8, 10), and it has been noted to extend into the early teens (4, 6, 9). Both acute respiratory illness episodes (4–8, 10) as well as occurrence of chronic

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Abbreviations: FEF_{25-75}, forced expiratory flow 25–75 per cent of forced vital capacity; FEF_{25-75}%, mean FEF_{25-75} per cent predicted; FEF-Z, FEF_{25-75}-Z score; FEV_1, forced expiratory volume in one second; FVC, forced vital capacity; NHLBI, National Heart, Lung and Blood Institute.

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symptoms (9, 11) have been reported. However, with the exception of a study by Schilling et al. (12) which failed to find any effect, the pulmonary function correlates of the exposure to parental smoking have been largely unexplored.

As part of a prospective study of risk factors for obstructive airways disease which may be operative early in life, we have investigated the effects of parents' smoking patterns on the pulmonary function of their children. Our findings suggest that a child's passive exposure to cigarette smoke has an adverse effect on the child's pulmonary function.

Materials and Methods

Selection of the sample

A random sample was selected from all children, aged 5–9-years-of-age, in the public and parochial schools of East Boston, Massachusetts, as of September, 1974. Thirty-two per cent of all children in each school in the community studied were randomly chosen to ensure a uniform geographic distribution in the community. The number of children initially selected for this study was based on estimates of expected refusal rates and on the estimated number of children needed for a follow-up study on familial patterns of chronic productive cough symptoms and obstructive airways disease as determined from a previous study in this community (13). Table 1 details the outcome of the selected sample.

The community of East Boston is a geographically defined neighborhood within the city of Boston in which the inhabitants are of predominantly Italian-American descent. Sixty-three per cent of working adults in the present sample were employed as clerks (or in related clerical positions), craftsmen, service workers, or were among the operatives defined by the US Census definitions (14). Only 5 per cent held professional, technical or managerial positions. Approximately 40 per cent of the adults had at least a high school diploma.

Screening of the sample

Between January and June, 1975, interviewers visited the households of the children who had been selected for the random sample. These interviewers had been specially trained using materials provided by the Division of Lung Diseases, National Heart, Lung and Blood Institute (NHLBI). They enumerated all residents of the households and asked each to attend a special neighborhood clinic where we could obtain respiratory symptom and illness histories and measure of pulmonary function. Those families who agreed to participate but who did not come to the clinic were screened in their homes.

Standardized questionnaires were used to obtain histories of respiratory symptoms and illness as well as smoking histories and demographic data. Separate questionnaires were administered by the

Table 1

Outcome of sample of index children aged 5–9 years selected for study of the effect of parental smoking on their pulmonary function, East Boston, MA, 1974

Sample	No. of children
Selected	806
Not available*	116
Total living in study community	690
Not contacted after 3 home visits	50
Located	640
Parents refused	175 (27.3%)†
Language problem‡	5
Other	4
Total interviewed	456 (71.3%)†
Total with usable data	444 (69.4%)†

* The large proportion of persons who moved prior to being contacted can be attributed directly to a period of instability related to problems of school desegregation in the city of Boston.

† Per cent of 640.

‡ Did not speak English or Italian.

interviewers for subjects less than 10 years of age and those 10 years and older. Questions relating to chronic cough, phlegm and chest illness were those proposed for lung program epidemiology studies by the Division of Lung Diseases, NHLBI (15). Parents answered for children ages 10 years and younger for all questions except those pertaining to the child's smoking history; all other persons answered for themselves. Smoking histories were obtained from all children in the absence of their parents during the pulmonary function testing.

Pulmonary function testing

Subjects performed forced vital capacity (FVC) maneuvers while in the sitting position and without the use of a nose clip, using an eight liter, water-filled, portable, recording spirometer (Survey Spirometer, Warren Collins, Inc., Braintree, MA). Subjects were encouraged to perform FVC maneuvers until five acceptable tracings were obtained, or until it became evident that they could not perform adequately. A tracing was considered acceptable if it was at least four seconds in duration (adults were encouraged to blow for at least six seconds) and if the interviewer felt that a maximal effort had been made. All tests on children were done by one of two interviewers, each with at least two years of experience. Each subject's standing height was measured without shoes to the nearest one-half inch. All pulmonary function measurements were corrected to body temperature, pressure and water saturation.

One-second forced expiratory volume (FEV_1) and forced expiratory flow 25–75 (FEF_{25-75}) were obtained by standard techniques (16). The working FEV_1 was obtained as the mean from the best three of five tracings as recommended by the Division of Lung Diseases, NHLBI (15). FEF_{25-75}'s were obtained from the same tracings, and a mean was calculated. The working means were converted into per cent predicted values using the nomograms of Dickman et al. (17) for subjects less than age 25 years and those of Ferris et al. (18) for subjects 25 years and older.

In addition to the retrospective respiratory symptom and illness histories obtained at each subject's entrance into the study, the acute respiratory illness experience for the five through nine-year-old children was assessed prospectively over a two-year period using methods previously described (19). Briefly, parents of children were called by telephone every two weeks (except in July and August) for the two-year period September, 1975, through June, 1977. Those children who had experienced one or more selected respiratory symptoms in the previous two weeks were visited in their homes for a more detailed history of their respiratory symptoms. Definitions of upper and lower respiratory illness were identical to those proposed by Monto (20).

Definitions of cigarette smoking

Adults (age 20 years and older). Adults were defined as having never smoked if they never smoked or smoked less than one cigarette per day for more than one year, or less than 20 packs during their lifetime. Current smokers were defined as those who smoked more than these amounts and who were smoking within one month of the time of interview. Ex-smokers were persons who had stopped smoking more than one month before the time of interview and who had smoked more than the above amounts.

Children (age 19 years or younger). Children were considered to have never smoked if they never smoked as much as one cigarette per week. They were classified as ever-smokers if they were currently smoking or had at some time smoked as much as one cigarette per week.

Analysis of data

Households were divided into three groups on the basis of parental smoking

pattern (table 2), and only those for which interview data were available for both parents have been included in this analysis. Households in which only one parent was interviewed were excluded since, for the purposes of this analysis, it was necessary to have accurate smoking histories for both parents throughout the lifetime of the children in the household.

FEF_{25-75} was used in the present analysis because it provided better discrimination between children in the various household smoking groups than did FEV_1. Initial age-sex standardization was carried out using the nomograms referred to previously. However, it was observed that the variability around the mean FEF_{25-75} per cent predicted ($FEF_{25-75}\%$) was high (1 SD > 20%). Therefore, to decrease the variability of the $FEF_{25-75}\%$ measurement, and thereby increase the efficiency of the analysis and provide a more direct means of comparing children in different age-sex categories, an $FEF_{25-75}\%$ score (FEF-Z score) was derived as follows: Children were divided into sex-specific, five-year age groups, and adults divided into two sex-specific groups. Within each group, subjects were rank ordered and the ranks converted into a cumulative frequency distribution. Each rank was then assigned a score from a table of areas under a standard normal curve (21). Each score corresponded to the position of the rank in the cumulative frequency distribution. The mean score within each group was thus 0 with a variance of 1. The scores can, for example, be interpreted as follows (figure 1): persons with a score of +1 would have an $FEF_{25-75}\%$ equal to or greater than 84 per cent of the members of their age-sex spe-

TABLE 2

Classification of households on the basis of parental smoking pattern, East Boston, MA, 1974

Two "never" smoking parents (type 0)
a. Both parents never smoked.
or
b. One or both parents ex-smokers but neither smoked at any time during the lifetime of all children.
Two "current" smoking parents (type 2)
a. Both parents current smokers and smoked during first year of life of all children*
or
b. One or both parents ex-smokers but both smoked during first year of life of all children.
One "current" smoking parent (type 1)
a. One "never" and one "current" parent smoker, as defined above.

* No current smokers excluded on this basis.

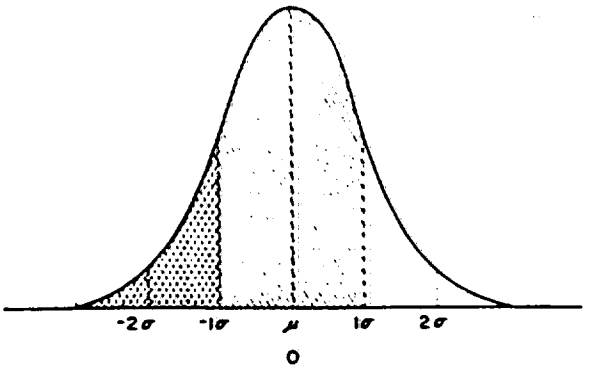

FIGURE 1. Interpretation of FEF-Z score. The FEF-Z scores are normally distributed with a mean (μ) of 0 and variance (σ^2) of 1. Subjects with a score of 1 (1σ) would have an $FEF_{25-75}\%$ predicted equal to or greater than 84 per cent of their peers (hatched + cross-hatched areas). Subjects with a score of -1 (1σ) would have an $FEF_{25-75}\%$ equal to or greater than only 16 per cent of their peers (cross-hatched area).

cific group; persons with a score of −1 would have an FEF_{25-75}% equal to or greater than only 16 per cent of their group. Linear regression (22, pp. 135–147) of FEF-Z score on age failed to show any trend toward age ordering of the ranks within each age-sex specific group for subjects aged 5–19 years (all except three children were age 19 years or younger).

A weighted mean FEF-Z was obtained for the children of each household included in the analysis. The weights were derived from a one-way analysis of variance (22, pp. 279–285) which had demonstrated familial similarity of level of FEF-Z score. The effect of the weighting procedure is to take into account the varying size of sibships in the calculation of the overall means (see Appendix).

All tests of statistical significance have been reported as one-sided *p* values. The use of one-sided *p* values was felt to be justified since the alternative hypothesis of a protective effect of parental smoking on pulmonary function of children did not seem plausible.

Selection of households for analysis

The 444 index children initially entered into the study (table 1) came from 404 households. In 318 of these households (79.0 per cent), both parents were living in the household at the time of survey. Two-hundred and forty-six of these 318 households (77.4 per cent) had complete data collected for both parents (in 70 households, the fathers refused to be interviewed, and in two, the mothers refused). These households where only one parent was interviewed were comparable to those where both parents were interviewed in terms of number of children per household interviewed (2.5 vs. 2.5), children's age in years (median, 8.4 vs. 8.4), and sex of children (per cent male, 52 vs. 53), per cent of children who had never smoked (19.6 per cent vs. 17.3 per cent), density of people in the household (persons per room, 0.84 vs. 1.00), type of heating system in the house (per cent gas, 33.8 vs. 29.3), and the level of education of the interviewed parents (per cent with high school diploma, 43.1 vs. 45.9).

Of the 246 households with data for both parents, 154 (62.6 per cent) had adequate smoking history and pulmonary function data for at least one child in the household. Ninety-two households where both parents were interviewed were excluded because no child in the household had data for both pulmonary function and smoking history. In table 3, the characteristics of the 92 excluded households are compared with those of the 154 included in the study. Children from excluded households were younger and came from households where fewer chil-

TABLE 3

Characteristics of East Boston, MA, households for which both parents were interviewed, January–June, 1975

Comparability factor	Included in present analysis	Excluded from present analysis
No. of households	154	92
Children household interviewed	3.06	1.45
% male children	57.4	56.3
Median age in years of children (range)	9.4 (2.5)*	6.4 (1.5)
Median persons/room (range)	1.00 (0.5–2.0)	0.80 (0.3–2.0)
% with gas heater	32.9	26.4
% of parents with high school diploma	35.6	45.4†

* Only three subjects older than age 19 years.

† Difference not significant

dren per household had been interviewed. In other respects, these households were comparable to those included in the analysis.

A further 29 per cent of the 445 children of all ages available from the 154 households which form the basis of this report had missing data for pulmonary function and/or smoking history and, therefore, could not be included in the present analysis. Children who were excluded for these reasons were younger (median age, 8 years; mean age, 8.4 ± 3.6 years) than children available for analysis (median age, 10 years, mean age, 10.3 ± 3.7 years) but were comparable with regard to sex distribution (per cent male, 54.4 vs. 52.2). There were 87 children who were excluded only because of missing pulmonary function data. Thirty-seven of these children from types 0 and 1 households (table 2) had smoking histories which were comparable to children included in the analysis (never, 81.1 per cent vs. 82.1 per cent; ever, 18.9 per cent vs. 17.9 per cent) and their median age (9 years) closely approximated that of children in the analysis. Fifty excluded children were from type 2 households and had a lower rate of personal cigarette smoking than those included in the analysis (ever, 12.0 per cent vs. 21.9 per cent). This is accounted for by the fact that 34 (68.0 per cent) of these 50 children were aged nine years or less and had never smoked (median age for all 50 children, 7.5 years). This rate of smoking compares with a rate of having ever-smoked of 6.6 per cent in the children aged nine years or less included in the analysis ($p = 0.124$, Fisher's Exact Test).

Results

Children from type 0 households (table 2) who themselves had never smoked cigarettes had higher average levels of FEF-Z scores than children from type 1 or type 2 households who never smoked (table 4, "all sibships"). Average FEF-Z scores differed by 0.156 and 0.355 standard deviation units between type 0 households and type 1 and 2 households, respectively. Although none of the specific differences are statistically significant (table 4), the trend is consistent with a decreasing level of function in the children with increasing parental smoking (χ^2_1 trend = 3.316; $p \sim 0.035$).

An identical result to that observed above is obtained if the analysis is restricted to children who never smoked and who also did not have any siblings who smoked (table 4, "sibships with only non-smoking children"). The comparable

TABLE 4

*Level of FEF-Z score for children who had never smoked, East Boston, MA, 1975**

	All sibships, by household type			Sibships with only non-smoking children, by household type			Sibships with siblings who smoked, by household type†	
	0	1	2	0	1	2	1	2
Average FEF-Z score	0.357‡	0.201§	0.002	0.357	0.194¶	0.046	0.222	−0.173
No. children	26	75	160	26	57	115	16	45
No. households	15	41	90	15	31	66	10	24

* Eight households contained only children who had ever smoked, and they are not included in this table.

† No children in type 0 households ever smoked and, therefore, type 0 households are not included under this heading.

‡ $t_{\infty} = 1.530$, $p < 0.05$ for difference 0.357 − 0.002. $t_{\infty} = 1.314$, $p < 0.20$ for difference 0.357 − 0.046.

§ $t_{\infty} = 0.627$, $p < 0.25$ for difference 0.357 − 0.201.

¶ $t_{\infty} = 0.630$, $p < 0.25$ for difference 0.357 − 0.194.

differences between the households are 0.163 and 0.311, respectively. Again, the specific differences are not statistically significant but a clearly consistent trend is observed (χ^2_1 trend = 2.002; p = 0.079). This second analysis was carried out since some children who reported that they had never smoked might have smoked and this would most likely occur in families where there were also children who had ever smoked (2). Such misclassification would serve to exaggerate differences between the different parent-smoking household categories. That this may have occurred to some extent is suggested by the observation that the average level of FEF-Z score was lower for children who never smoked and who lived with smoking siblings at least in type 2 households (table 4, "sibships with siblings who smoked").

Compared to children who had never smoked, children who had ever smoked had lower levels of pulmonary function regardless of whether one or both parents were current smokers (table 5). In type 2 households, the FEF-Z score for smoking children was 0.355 standard deviation units lower than that for non-smoking children (−0.309 vs. 0.046, t_{51} = 1.797, p = 0.035). A similar comparison for type 1 households gave a difference of 0.429 standard deviation units (−0.235 vs. 0.194, t_{42} = 1.533, p = 0.065). When smoking children were compared to non-smoking children who came from type 0 households, mean FEF-Z scores differed by 0.666 (−0.309 vs. 0.357, t_{40} = 2.432, p = 0.01) and 0.592 (−0.235 vs. 0.357, t_{28} = 1.863, p = 0.035) standard deviation units for type 2 and type 1 households, respectively.

The possibility that the lower levels of FEF-Z scores in children of smoking parents were the result of an increased burden of respiratory illness was investigated. At entrance into the study, parents of children aged 5–9 years were asked if their child had ever had a doctor's diagnosis of acute bronchitis, pneumonia, croup or bronchiolitis, and the age at which each was first diagnosed (table 6). There was no consistent tendency for the respiratory illness burden to increase with increasing parental cigarette smoking, although the number of type 0 households is too small for a definitive statement.

TABLE 5

Level of FEF-Z score for children who had ever smoked by household type, East Boston, MA, 1975

	Household type*	
	1	2
Average FEF-Z score	−0.235	−0.309
No. children	20	37
No. households	13	27

* There were no children who ever smoked in type 0 households

TABLE 6

Respiratory illness reported to have occurred prior to entrance into the study for East Boston, MA, children aged 5–9 years†, as reported at interview of parents, January–June, 1975*

	Household type		
	0	1	2
1st illness before age 1 year	1 (8.3)‡	8 (21.6)‡	7 (8.5)‡
No. households	12	37	82

* Acute bronchitis, pneumonia, croup and bronchiolitis reported in response to the question: "Has a doctor ever told you that _____ had _____?"

† All children aged 5–9 years in the various household groups for whom such data were available are included, regardless of whether or not they were included in the analysis of pulmonary function.

‡ Per cent

Further confirmation of a lack of association of parents' smoking and respiratory illness frequency is found in table 7. The cumulative two-year respiratory illness frequency as determined directly in a prospective study was very similar for children in the three parent smoking categories. Moreover, there was no trend toward increasing illness burden with an increasing number of parents who smoked.

Indices of household crowding and type of home heating system were also measured. The mean number of persons per room was virtually identical for the three household types (type 0 = 1.04, type 1 = 1.10, type 2 = 1.00). Analysis of the number of homes with central gas heating systems in kitchens (a common type of home heating system in East Boston) showed that type 2 households had a smaller percentage of such systems (23.7 per cent) than type 0 (54.5 per cent) and type 1 households (78.3 per cent).

Attempts to directly monitor the indoor environments of the three parent smoking groups for levels of various air pollutants were unsuccessful because we were unable to obtain the cooperation of a sufficient number of households. Data from previous work in East Boston (13) have shown that there are no overall differences in outdoor air quality in various parts of the community.

Discussion

The data presented in this report indicate that the cigarette smoking habits of parents may have a measurable effect on the pulmonary function of their non-smoking and smoking children. The effect is predominantly due to parental smoking and is not due to the confounding effect of the children's smoking habits. The average level of age-, height- and sex-standardized FEF_{25-75} of children who had never smoked declined progressively between families with two parents who had never smoked through families with two currently smoking parents (table 4). The average decline in FEF_{25-75} ranged between 0.15–0.20 standard deviation units with the addition of each smoking parent to the household. Moreover, the difference in the average level of FEF_{25-75} was as large between non-smoking children with two non-smoking parents (type 0 households) and similar children with two smoking parents and no smoking siblings (0.311 standard deviation units) as it was between smoking and non-smoking children with two smoking parents (type 2 households, 0.355 standard deviation units).

Although some of the specific comparisons between the parent-smoking household groups did not achieve statistical significance, the patterns of decline in

Table 7

Total two-year respiratory illness experience for children aged 5–9 years, by parent smoking category, East Boston, MA, 1975–1977*

No. of illness(es)	Parent smoking category		
	Type 0	Type 1	Type 2
0–1	2(8.7)†	17(21.3)	20(21.3)
2–3	7(30.4)	31(38.8)	31(33.0)
>3	14(60.9)	32(40.0)	43(45.7)
Total	23	80	94

* Includes upper and lower respiratory illnesses and episodes of isolated cough (20). All children aged 5–9 years in the various household groups for whom such data were available are included, regardless of whether or not they were included in the analysis of pulmonary function.

† Per cent of column total—males and females combined because their rates were not significantly different ($\chi^2_1 = 3.920$; $p > 0.417$).

levels of the standardized FEF_{25-75} were consistent in all the analyses. Those comparisons which reflected the extremes of what might be considered a dose-response relationship between parental smoking and the children's level of FEF_{25-75} (e.g., non-smoking children of two non-smoking parents vs. smoking children of two currently smoking parents) did achieve statistical significance.

The question of the validity of the observations reported bears directly on the nature of the sample of subjects used in the present analysis and the method for standardizing FEF_{25-75}. Families excluded because only one parent was interviewed were comparable to families with two parents interviewed with regard to important demographic and smoking variables. A potential problem does exist in the 154 two-parent families finally selected for analysis. Children from these families who were excluded due to missing pulmonary function and/or smoking history data were younger and, consequently, smoked less than those used in the analysis. Most of these differences were accounted for by the children excluded in the type 2 families. These excluded children could have biased the results only if their relative youth and lack of smoking experience diminished or enhanced their susceptibility to the effects of their parents' smoking. There is no evidence to suggest the former possibility, nor does it seem likely. Any enhanced susceptibility which these excluded type 2 children might show would tend to increase the strength of the reported associations. Therefore, our results could be biased toward an underestimate of the true magnitude of the observed relationship.

The use of the FEF-Z score is not likely to account for the observed results. Preliminary analysis demonstrated that there was no age ordering of the scores either within each age group or in the entire group aged 5–19 years (i.e., the group that included all but three of the children in this analysis). Thus, any minor age differences between the three household groups could not account for the findings in this study. Although the use of the FEF-Z score does not permit a direct conversion back to a flow rate, this is not a serious limitation for the present report. The major interest here is in relative differences between groups of children. For this purpose, the score is ideal since it allows estimates of relative differences across a wide range of ages in terms of standard devintion units which are easily understandable.

Our findings differ from those of Schilling et al. (12), who addressed this problem but were unable to demonstrate any effect of parental smoking on the pulmonary function of children. Several factors might explain this variation. The definitions of parental smoking differed, with Schilling et al. (12) using only parents' current smoking as the criterion. Different measures of pulmonary function are also reported, and the disparity in the two sets of findings may reflect differences in the inherent ability of various measures of pulmonary function to distinguish between the groups of children. Finally, the analysis of variance used by Schilling et al. does not appear to properly correct for the differences in sibship size and the fact that there is a significant intraclass correlation of pulmonary function among siblings. These factors have been controlled in the present analysis.

The mechanisms that underlie the progressive decline in FEF_{25-75} with increasing parental smoking are conjectural. Our data do not suggest that an increased respiratory illness burden is the explanation, as has been suggested by others (7, 8, 10). Studies which have examined children in the first few years of life have consistently found an association between parental cigarette smoking and increased episodes of respiratory illness (7, 8, 10). The present data are retrospective in this

regard (table 6), and the number of type 0 households available for analysis was very small. Therefore, our data may not be as sensitive as those in studies which directly observed infants and very young children. Studies using populations of children comparable in age to our own have focused largely on the relationship of chronic symptoms (5, 9, 11) to smoking habits of adults, and their data are not comparable to ours. In one of these studies (5), which evaluated acute respiratory tract illness, data were collected retrospectively with no confirmation by prospective follow-up. A series of studies (4, 6), which focused on acute respiratory illness in relation to parental smoking, did show an association between an increased number of such illnesses in the children of smoking parents in the seven days preceding interview. It is difficult to compare such findings with our own, since the methods and period of observation are so different.

Although the assessment of socioeconomic status and individual household environment is incomplete, our limited data do not suggest that differences in these variables play a major role in explaining the results we obtained. Indices of crowding were comparable in the three household groups. Type 2 households were least likely to have large gas-burning heating units in their homes. Finally, the range of socioeconomic status, as indicated by job and level of education, is limited in the study community. Because of the small number of type 0 households, a meaningful analysis of these factors could not be undertaken.

The possibility that contamination of indoor air by cigarette smoke may play a part in our findings has not been systematically explored. We were unable to determine satisfactorily to what extent the atmospheres of the homes of the various household groups differed. However, others (3, 23, 24) have demonstrated that cigarette smoke contaminates indoor air to a substantial extent and that substantial amounts of respirable particulates such as tar and nicotine, as well as carbon monoxide and other gases can be detected. The mechanisms by which this contamination might affect the lungs of non-smokers have not been well studied. Tars have been shown to be allergens capable of inducing precipitin reactions in rabbits (25), and, although it is not believed to be an allergen itself, nicotine may be able to function as a hapten when combined with other substances (26). Becker et al. (27, 28) have isolated a glycoprotein present in tobacco smoke to which human volunteers demonstrate cutaneous hypersensitivity and which is capable of activating Factor XII (Hagerman factor). They postulate that the glycoprotein may trigger an inflammatory response via activation of Factor XII which might be related to the pulmonary injury associated with cigarette use. Furthermore, the amount of this antigen in smoke is such that it may be capable of exerting its effects in adjacent non-smokers as well. Surely, a great deal more work will be needed to clearly define the risks to the lungs of non-smokers.

At present, obviously, it is impossible to know the long-range health effects of the observations reported here regarding the passive effects of parental smoking. The long-range effects are the focus of the prospective portion of this study as it continues over time.

In addition, although this report has focused on the passive effects of parental smoking, it is important to remember the very direct effect of the children's own smoking habits on their relative levels of FEF_{25-75}. These data lend additional support to observations made in teenagers (29, 30) that cigarette smoking in children has measurable functional consequences despite limited daily and total cigarette consumption. Additional data

are being collected in the East Boston population to explore this problem in more depth.

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Appendix

A. *One-Way Analysis of Variance-Random Effects Model*

1. $Y_{ij} = \mu + A_i + e_{ij}; A_i \sim N(0, \sigma_A^2), e_{ij} \sim N(0, \sigma^2)$,
where Y_{ij} = FEF-Z score for the jth person in the ith household,
μ = mean FEF-Z score for the population,

A_i = random effect due to family, and
e_{ij} = random effect for jth person in ith family.

2. $Y_i = \mu + A_i + \bar{e}_i$,
where Y = the average FEF-Z score for the ith family,
A_i = as above, and
$\bar{e}_i$ = random effect within the ith family and = $\Sigma e_{ij}/N_i$
and variance $(Y_i) = \sigma_1^2 + \sigma^2/N_i$
where N_i = number of persons in ith family.

3. $\bar{\bar{Y}} = \Sigma w_i Y_i/\Sigma w_i$
where $w_i = 1/(\sigma_1^2 + \sigma^2/N_i)$
and variance $(\bar{\bar{Y}}) = 1/\Sigma w_i$

B. *Example of Weighting Procedure*

From 1 Way ANOVA $\sigma^2 = 0.723$ $\sigma_1^2 = 0.230$

Family	No. in ith Family	$\bar{Y}_i$	w_i	$w_i Y_i$
1	1	1.889	1.049	1.982
2	2	−0.191	1.691	−0.323
3	3	1.255	2.123	2.664
			$\Sigma w_i = 4.863$	$\Sigma w_i \bar{Y}_i = 4.323$

$$\bar{\bar{Y}} = \frac{4.323}{4.863} = 0.889$$